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>>B7.D763,D570,D575,D380,D283,D285,D28

>>"1".D763,D570,D575,D380,D283,D285,D2
>>1S.D763,D570,D575,D380,D283,D285,D28
>>PSORIASIS.D763,D570,D575,D380,D283,I
>>PSORIASI.D763,D570,D575,D380,D283,D
>>(PSORIASIS AND (B7 OR (B7 AD.

Results -

289,D291,D193,D194,D195,D196,D197,D198,D199,
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289,D291,D193,D194,D195,D196,D197,D198,D199
289,D291,D193,D194,D195,D196,D197,D198,D199,
,D287,D289,D291,D193,D194,D195,D196,D197,D1
D287,D289,D291,D193,D194,D195,D196,D197,D19
'))).P28-P85,P23-P27,P20-P22,P1-P18.=135

Database: US Patents Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Refine Search: (b7 or b7 adj 1) and soriasis) Clear

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USPT,JPAB,EPAB,DWPI	(b7 or b7 adj 1) and	toimmun$ or psoriasis)	429	L1

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7. miller et al. immunological reviews 144 : 225 -244 (1995)
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3. tamada et al. annals of allergy asthma and immunology 85 / 3 : 164 - 174 (2000)
4. salojin et al. immunology today 19 / 10 : 468 - 473 (1998)
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8. anderson et al. current opinion in immunology 11 / 6 : 677 - 683 (2000) ****
9. krensky et al. current opinion in investigational drugs 5 / 7 : 809 - 818 !(996) (peptides)
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3. tamada et al. annals of allergy asthma and immunology 85 / 3 : 164 - 174 (2000)
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3. salojin et al. immunology today 19 / 10 : 468 - 473 (!998)
4. sfikakis et al. clinical rheumatology 18 / 4 : 317 - 327 (1999)
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3. tamada et al. annals of allergy asthma and immunology 85 / 3 : 164 - 174 (2000)
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Volume 94, Number 2, February 2000

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4. nickoloff et al. journal of cutaneous pathology 19 / 6 : page 543 (1992)

5. nickoloff et al. journal of immunology 150 / 6: 2148 - 2159 (!993)

6. nickoloff et al. blood 83 / 9 : 2580 - 2586 (1994)

7. denfeld et al. european journal of immunology 26 / 10 : 2329 -2334 (1996) cd40

8. abrams et al. journal of clinical investigation 103 / 9 : 1243 - 1252 (1999)

9. gottlieb et al. journal of investigative dermatology 114 / 4 : page 840 (2000) ******

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3. salojin et al. immunology today 19 / 10 : 468 - 473 (!998)
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9. linsley et al. j. clin. invest. 95 / 6 : 2429- 2430 (1995)

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Article title	CD28/B7 regulation of autoimmune diabetes
Article identifier	0257277X9700037X
Authors	Herold_K_C Lenschow_D_J Bluestone_J_A
Journal title	Immunologic Research
ISSN	0257-277X
Publisher	Humana Press
Year of publication	1997
Volume	16
Issue	1
Supplement	0
Page range	71-84
Number of pages	14
User name	Adonis
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8. abrams et al. journal of clinical investigation 103 / 9 : 1243 - 1252 (1999)

9. gottlieb et al. journal of investigative dermatology 114 / 4 : page 840 (2000) ******

10. djukanovic et al. clinical and experimental allergy 30 supplement 1 : 46 - 50 (2000)

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Article title	Animal models of psoriasis - What can we learn from them?
Article identifier	0022202X99110157
Authors	Schön_M_P
Journal title	Journal of Investigative Dermatology
ISSN	0022-202X
Publisher	Blackwell USA
Year of publication	1999
Volume	112
Issue	4
Supplement	0
Page range	405-410
Number of pages	6
User name	Adonis
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Article title	The role of co-stimulation in airway inflammation
Article identifier	0960217800106535
Authors	Djukanovic_R
Journal title	Clinical and Experimental Allergy, Supplement
ISSN	0960-2178
Publisher	Blackwell UK
Year of publication	2000
Volume	30
Issue	S1
Supplement	1
Page range	46-50
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9. gottlieb et al. journal of investigative dermatology 114 / 4 : page 840 (2000) ******

10. djukanovic et al. clinical and experimental allergy 30 supplement 1 : 46 - 50 (2000)

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Article title	Abstracts: 536
Article identifier	0022202X00112126
Authors	
Journal title	Journal of Investigative Dermatology
ISSN	0022-202X
Publisher	Blackwell USA
Year of publication	2000
Volume	114
Issue	4
Supplement	0
Page range	839-868
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Article title	Evolution of the T-Cell Repertoire during the Course of Experimental Immune-Mediated Demyelinating Diseases
Article identifier	0105289695100678
Authors	Miller_S_D McRae_B_L Vanderlugt_C_L Nikcevich_K_M Pope_J_G Pope_L Karpus_W_J
Journal title	Immunological Reviews
ISSN	0105-2896
Publisher	Munksgaard
Year of publication	1995
Volume	144
Issue	1
Supplement	0
Page range	225-244
Number of pages	20
User name	Adonis
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8. abrams et al. journal of clinical investigation 103 / 9 : 1243 - 1252 (1999)

9. gottlieb et al. journal of investigative dermatology 114 / 4 : page 840 (2000) ******

10. djukanovic et al. clinical and experimental allergy 30 supplement 1 : 46 - 50 (2000)

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STIC-ILL MIC

From: Gambel, Phillip
Sent: Sunday, February 18, 2001 8:43 AM
To: STIC-ILL
Subject: 09/383916 b7 and psoriasis

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1644 mailbox

1. schon et al. journal of investigative dermatology 112 / 4 : 405 - 410 (1999)

2.wallace et al. j. clinical rheumatol. 6 /1 : 49 - 54 (2000)

3. najafian et al. expert opinion on investigational drugs 9 / 9 : 2147 - 2157 (2000)

4. nickoloff et al. journal of cutaneous pathology 19 / 6 : page 543 (1992)

5. nickoloff et al. journal of immunology 150 / 6: 2148 - 2159 (!993)

6. nickoloff et al. blood 83 / 9 : 2580 - 2586 (1994)

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8. abrams et al. journal of clinical investigation 103 / 9 : 1243 - 1252 (1999)

9. gottlieb et al. journal of investigative dermatology 114 / 4 : page 840 (2000) ******

10. djukanovic et al. clinical and experimental allergy 30 supplement 1 : 46 - 50 (2000)

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From: Gambel, Phillip
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To: STIC-ILL
Subject: b7 and cd28 and autoimmunity

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1. daikh et al. journal of leukocyte biology 62 / 2 : 156 - 162 (1997)
2. harlan et al. clinical immunology and immunopathology 75 / 2 : 99 - 111 (1995)
3. tamada et al. annals of allergy asthma and immunology 85 / 3 : 164 - 174 (2000)
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6. kobata et al. reviews in immunogenetics 2 /1 : 74 ??? - 80 (2000)
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8. anderson et al. current opinion in immunology 11 / 6 : 677 - 683 (2000) ****
9. krensky et al. current opinion in investigational drugs 5 / 7 : 809 - 818 !(996) (peptides)
10. new england journal of medicine 335 / 18 : 1369 - 13777 (1996)

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current opinion in urology

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Sent: Sunday, February 18, 2001 9:01 AM
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1. daikh et al. journal of leukocyte biology 62 / 2 : 156 - 162 (1997)
2. harlan et al. clinical immunology and immunopathology 75 / 2 : 99 - 111 (1995)
3. tamada et al. annals of allergy asthma and immunology 85 / 3 : 164 - 174 (2000)
4. salojin et al. immunology today 19 / 10 : 468 - 473 (1998)
5. diehl et al. journal of molecular medicine 78 / 7 : 363 - 371 (2000) CD40
6. kobata et al. reviews in immunogenetics 2 /1 : 74 ??? - 80 (2000)
7. Christadoss et al. clinical immunolgoy 94 / 2 : 75 ??? - 87 (2000)
8. anderson et al. current opinion in immunology 11 / 6 : 677 - 683 (2000) ****
9. krensky et al. current opinion in investigational drugs 5 / 7 : 809 - 818 !(996) (peptides)
10. new england journal of medicine 335 / 18 : 1369 - 13777 (1996)

thanx

current opinion in urology

Article title	The role of CD40 in peripheral T cell tolerance and immunity
Article identifier	0946271600105936
Authors	Diehl_L Den_Boer_A_T Van_der_Voort_E_I_H Melief_C_J_M Offringa_R Toes_R_E_M
Journal title	Journal of Molecular Medicine
ISSN	0946-2716
Publisher	Springer
Year of publication	2000
Volume	78
Issue	7
Supplement	0
Page range	363-371
Number of pages	9
User name	Adonis
Cost centre	
PCC	$20.00
Date and time	Thursday, February 22, 2001 12:49:08 AM

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et     Items   Description
;1         6   AU="ANDERSON DARRELL R"
;2         6   RD S1 (unique items)
;3         6   (16C10 OR 7C10 OR 20C9 OR 7B6) AND (B7 OR B7(W)1)
;4         2   RD S3 (unique items)
;5        90   (B7 OR B7(W)1) AND PSORIASIS
;6        51   RD S5 (unique items)
;7       966   (B7 OR B7(W)1) AND AUTOIMMUN?
;8       264   S7 AND (SCLEROSIS OR DIABETES)
;9        21   S8 AND REVIEW?
;10       15   RD S9 (unique items)
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           966  S7
       2851120  REVIEW?
    S11     84  S7 AND REVIEW?
? rd s11

...examined 50 records  (50)
...completed examining records
    S12     62  RD S11 (unique items)
? ds


Set     Items   Description
S1          6   AU="ANDERSON DARRELL R"
S2          6   RD S1 (unique items)
S3          6   (16C10 OR 7C10 OR 20C9 OR 7B6) AND (B7 OR B7(W)1)
S4          2   RD S3 (unique items)
S5         90   (B7 OR B7(W)1) AND PSORIASIS
S6         51   RD S5 (unique items)
S7        966   (B7 OR B7(W)1) AND AUTOIMMUN?
S8        264   S7 AND (SCLEROSIS OR DIABETES)
S9         21   S8 AND REVIEW?
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S11        84   S7 AND REVIEW?
S12        62   RD S11 (unique items)
? t s12/3/all


 12/3/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

12181396   BIOSIS NO.: 199900476245
Differential requirements of naive and memory T cells for CD28
  costimulation in autoimmune pathogenesis.
AUTHOR: Perrin P J(a); Lovett-Racke A; Phillips S M; Racke M K
AUTHOR ADDRESS: (a)Department of Medicine, University of Pennsylvania, 909
  BRB II, 421 Curie Boulevard, Philadelphia, PA, 19104-6160**USA
JOURNAL: Histology and Histopathology  14 (4):p1269-1276 Oct., 1999
ISSN: 0213-3911
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
```

12/3/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12150299 BIOSIS NO.: 199900445148
Multiple sclerosis : Pathology and pathogenesis.
AUTHOR: Prasad Rameshwar(a); Prasad Roli(a); Wacek Bartholomew(a); Chandran Rajeswari(a); Ilangovan Saroja(a)
AUTHOR ADDRESS: (a)Department of Pathology, University of Illinois at Chicago, 1819 West Polk Street, Chicago, IL, 60612-7335**USA
JOURNAL: Proceedings of the National Academy of Sciences India Section B (Biological Sciences) 68 (3-4):p189-198 1998
ISSN: 0369-8211
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

12/3/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11729727 BIOSIS NO.: 199800511458
Endothelial cells as antigen-presenting cells: Role in human transplant rejection.
AUTHOR: Rose M L(a)
AUTHOR ADDRESS: (a)National Heart Lung Inst., Imperial Coll. Sch. Med., Heart Sci. Centre, Harefield Hosp., Harefie**UK
JOURNAL: CMLS Cellular and Molecular Life Sciences 54 (9):p965-978 Sept., 1998
ISSN: 1420-682X
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

12/3/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11669616 BIOSIS NO.: 199800451347
Targeting the **B7** /CD28: CTLA-4 costimulatory system in CNS **autoimmune** disease.
AUTHOR: Karandikar Nitin J; Vanderlugt Carol L; Bluestone Jeffrey A; Miller Stephen D(a)
AUTHOR ADDRESS: (a)Dep. Microbiol.-Immunol. Interdepartmental Immunobiol. Cent., North Western Univ. Med. Sch., 303**USA
JOURNAL: Journal of Neuroimmunology 89 (1-2):p10-18 Aug. 14, 1998
ISSN: 0165-5728
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

12/3/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11117657 BIOSIS NO.: 199799738802
The CD28-**B7** costimulatory pathway and its role in **autoimmune** disease.
AUTHOR: Daikh David; Wofsy David; Imboden John B(a)
AUTHOR ADDRESS: (a)Box 0868, Univ. California, San Francisco, CA 94143**USA
JOURNAL: Journal of Leukocyte Biology 62 (2):p156-162 1997

SSN: 0741-5400
)OCUMENT TYPE: Literature Review
(ECORD TYPE: Abstract
.ANGUAGE: English

12/3/6 (Item 6 from file: 5)
IALOG(R)File 5:Biosis Previews(R)
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9812931 BIOSIS NO.: 199598267849
)tential roles of the **B7** and CD28 receptor families in **autoimmunity** and immune evasion.
THOR: Harlan David M(a); Abe Ryo; Lee Kelvin P; June Carl H
THOR ADDRESS: (a)Immune Cell Biol. Program, Naval Med. Res. Inst., Bethesda, MD 20889**USA
)URNAL: Clinical Immunology and Immunopathology 75 (2):p99-111 1995
;SN: 0090-1229
)CUMENT TYPE: Literature Review
:CORD TYPE: Abstract
\NGUAGE: English

2/3/7 (Item 1 from file: 73)
7\LOG(R)File 73:EMBASE
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994642 EMBASE No: 2001039391
Mast cells as initiators of immunity and host defense
Henz B.M.; Maurer M.; Lippert U.; Worm M.; Babina M.
Prof. Dr. B.M. Henz, Department of Dermatology, Charite, Campus Virchow, Humboldt University, Augustenburgerplatz 1, 13344 Berlin Germany
AUTHOR EMAIL: magdalena.fuchs@@@charite.de
Experimental Dermatology (EXP. DERMATOL.) (Denmark) 2001, 10/1 (1-10)
CODEN: EXDEE ISSN: 0906-6705
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 102

2/3/8 (Item 2 from file: 73)
ALOG(R)File 73:EMBASE
) 2001 Elsevier Science B.V. All rts. reserv.

)993861 EMBASE No: 2001038222
T lymphocyte costimulatory molecules in host defense and immunologic seases
Tamada K.; Chen L.
Dr. L. Chen, Department of Immunology, Guggenheim 3, Mayo Clinic, 200 First St SW, Rochester, MN 55905 United States
Annals of Allergy, Asthma and Immunology (ANN. ALLERGY ASTHMA IMMUNOL.) 'nited States) 2000, 85/3 (164-174)
CODEN: ALAIF ISSN: 1081-1206
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 140

2/3/9 (Item 3 from file: 73)
ALOG(R)File 73:EMBASE
·) 2001 Elsevier Science B.V. All rts. reserv.

·927362 EMBASE No: 1998331678
T-cell anergy and altered t-cell receptor signaling: Effects on **\toimmune** disease
Salojin K.V.; Zhang J.; Madrenas J.; Delovitch T.L.

K.V. Salojin, Autoimmunity/Diabetes Group, Dept of Microbiology, University of Western Ontario, London, Ont. N6A 5K8 Canada
AUTHOR EMAIL: del@rri.on.ca
Immunology Today (IMMUNOL. TODAY) (United Kingdom) 1998, 19/10 (468-473)
CODEN: IMTOD ISSN: 0167-5699
PUBLISHER ITEM IDENTIFIER: S0167569998013267
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 71

2/3/10 (Item 4 from file: 73)
ALOG(R)File 73:EMBASE
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352352 EMBASE No: 2000333878
'he role of CD40 in peripheral T cell tolerance and immunity
)iehl L.; Den Boer A.T.; Van der Voort E.I.H.; Melief C.J.M.; Offringa R.
'oes R.E.M.
.. Diehl, Dept. Immunohematol. Blood Transfus., Medical Center, Leiden)niversity, Albinusdreef 2, 2333 ZA Leiden Netherlands
Tournal of Molecular Medicine (J. MOL. MED.) (Germany) 2000, 78/7 (363-371)
CODEN: JMLME ISSN: 0946-2716
)OCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 75

2/3/11 (Item 5 from file: 73)
\LOG(R)File 73:EMBASE
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344662 EMBASE No: 2000320786
Restricted usage of T-cell receptor Vgamma-Vdelta genes and expression of stimulatory molecules in Takayasu's arteritis
Seko Y.; Takahashi N.; Tada Y.; Yagita H.; Okumura K.; Nagai R.; Virgin; Irma; Miyazawa; Numano; Tanaka; Kimura
Y. Seko, Dept. of Cardiovascular Medicine, Graduate School of Medicine,)niversity of Tokyo, Tokyo 113-8655 Japan
\UTHOR EMAIL: sekoyosh-tky@umin.ac.jp
International Journal of Cardiology (INT. J. CARDIOL.) (Ireland) 31 \UG 2000, 75/SUPPL. 1 (S77-S83+S85-S87)
CODEN: IJCDD ISSN: 0167-5273
PUBLISHER ITEM IDENTIFIER: S0167527300001947
)OCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 33

2/3/12 (Item 6 from file: 73)
\LOG(R)File 73:EMBASE
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'35208 EMBASE No: 2000214674
'he genetics of Hashimoto's disease
Barbesino G.; Chiovato L.
)r. G. Barbesino, Department of Endocrinology, University of Pisa, Via 'aradisa 2, Pisa I-56124 Italy
\UTHOR EMAIL: zipeppe@tin.it
Endocrinology and Metabolism Clinics of North America (ENDOCRINOL. METAB. CLIN. NORTH AM.) (United States) 2000, 29/2 (357-374)
'ODEN: ECNAE ISSN: 0889-8529
'OCUMENT TYPE: Journal; Review
ANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

UMBER OF REFERENCES: 114

/3/13 (Item 7 from file: 73)
LOG(R)File 73:EMBASE
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35202 EMBASE No: 2000214668
he genetics of Graves' disease
ough S.C.L.
r. S.C.L. Gough, Division of Medical Sciences, Birmingham Heartlands
ospital, Bordesley Green East, Birmingham B9 5SS United Kingdom
JTHOR EMAIL: s.c.gough@bham.ac.uk
ndocrinology and Metabolism Clinics of North America (ENDOCRINOL.
ETAB. CLIN. NORTH AM.) (United States) 2000, 29/2 (255-266)
)DEN: ECNAE ISSN: 0889-8529
)CUMENT TYPE: Journal; Review
ANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
JMBER OF REFERENCES: 53

/3/14 (Item 8 from file: 73)
LOG(R)File 73:EMBASE
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31840 EMBASE No: 2000141343
olecular aspects in the pathogenesis of human systemic lupus
thematosus
iossis S.-N.C.; Tsokos G.C.
r. G.C. Tsokos, Department of Clinical Physiology, Walter Reed Army
nst. of Research, Bldg. 40, Washington, DC 20307-5100 United States
JTHOR EMAIL: gtsokos@usa.net
rchivum Immunologiae et Therapiae Experimentalis (ARCH. IMMUNOL. THER.
XP.) (Poland) 2000, 48/1 (11-19)
)DEN: AITEA ISSN: 0004-069X
)CUMENT TYPE: Journal; Review
ANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
JMBER OF REFERENCES: 57

/3/15 (Item 9 from file: 73)
LOG(R)File 73:EMBASE
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58832 EMBASE No: 2000134072
rimary biliary cirrhosis: An orchestrated immune response against
thelial cells
ershwin M.E.; Ansari A.A.; Mackay I.R.; Nakanuma Y.; Nishio A.; Rowley
.; Coppel R.L.
.E. Gershwin, Division of Rheumatology/Allergy, Univ. California School
f Medicine, TB 192, Davis, CA 95616 United States
JTHOR EMAIL: megershwin@ucdavis.edu
mmunological Reviews (IMMUNOL. REV.) (Denmark) 2000, 174/- (210-225)
)DEN: IMRED ISSN: 0105-2896
)CUMENT TYPE: Journal; Review
ANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

/3/16 (Item 10 from file: 73)
OG(R)File 73:EMBASE
2001 Elsevier Science B.V. All rts. reserv.

)3618 EMBASE No: 2000119397
ie role of costimulatory molecules as targets for new immunosuppressives
:ransplantation
.shimoto K.; Dong V.M.; Sayegh M.H.

H. Sayegh, Lab. Immunogenetic Transprantation, Brigham and Women's
)spital, 75 Francis Street, Boston, MA, 02115 United States
)THOR EMAIL: msayegh@rics.bwh.harvard.edu
ırrent Opinion in Urology (CURR. OPIN. UROL.) (United Kingdom) 2000,
)/2 (57-62)
)DEN: CUOUE ISSN: 0963-0643
)CUMENT TYPE: Journal; Review
\NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
)MBER OF REFERENCES: 49

'3/17 (Item 11 from file: 73)
.OG(R)File 73:EMBASE
2001 Elsevier Science B.V. All rts. reserv.

30656 EMBASE No: 2000096021
)le of costimulatory molecules in **autoimmunity**
)bata T.; Azuma M.; Yagita H.; Okumura K.
Kobata, Division of Immunology, Institute for Medical Science, Dokkyo
ıiversity, 880 Kitakobayashi, Mibu, Tochigi 321-0293 Japan
)THOR EMAIL: tkobata@dokkyomed.ac.jp
:views in Immunogenetics (REV. IMMUNOGEN.) (Denmark) 2000, 2/1
'4-80)
)DEN: RVIMF ISSN: 1398-1714
)CUMENT TYPE: Journal; Review
\NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
)MBER OF REFERENCES: 66

'3/18 (Item 12 from file: 73)
.OG(R)File 73:EMBASE
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8850 EMBASE No: 2000084275
ımune response and immunopathology of the inner ear: An update
ırcia Berrocal J.R.; Ramirez-Camacho R.
.R. Garcia Berrocal, Servicio de Otorrinolaringologia, Clinica Puerta de
.erro, San Martin de Porres 4, 28035 Madrid Spain
)urnal of Laryngology and Otology (J. LARYNGOL. OTOL.) (United Kingdom
2000, 114/2 (101-107)
)DEN: JLOTA ISSN: 0022-2151
)CUMENT TYPE: Journal; Review
\NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
)MBER OF REFERENCES: 36

'3/19 (Item 13 from file: 73)
.OG(R)File 73:EMBASE
2001 Elsevier Science B.V. All rts. reserv.

:1396 EMBASE No: 2000045784
ıimal models of myasthenia gravis
ıristadoss P.; Poussin M.; Deng C.
Christadoss, Dept. of Microbiology and Immunology, University of Texas
:dical Branch, Galveston, TX 77555-1070 United States
.inical Immunology (CLIN. IMMUNOL.) (United States) 2000, 94/2
'5-87)
)DEN: CLIIF ISSN: 1521-6616
)CUMENT TYPE: Journal; Review
\NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
)MBER OF REFERENCES: 109

'3/20 (Item 14 from file: 73)
.OG(R)File 73:EMBASE
2001 Elsevier Science B.V. All rts. reserv.

;1079 EMBASE No: 2000025912
)lecular pathogenesis of multiple sclerosis
ır-Or A.; Oliveira E.M.L.; Anderson D.E.; Hafler D.A.
A. Hafler, Center for Neurologic Diseases, Brigham and Women's
)spital, Harvard Medical School, 77 Avenue Louis Pasteur, Boston, MA
:115-5187 United States
)THOR EMAIL: hafler@cnd.bwh.harvard.edu
)urnal of Neuroimmunology (J. NEUROIMMUNOL.) (Netherlands) 1999,
)0/1-2 (252-259)
)DEN: JNRID ISSN: 0165-5728
)BLISHER ITEM IDENTIFIER: S0165572899001939
)CUMENT TYPE: Journal; Review
\NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
)MBER OF REFERENCES: 96

'3/21 (Item 15 from file: 73)
)OG(R)File 73:EMBASE
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;1093 EMBASE No: 1999415701
ıe **B7**-CD28/CTLA-4 costimulatory pathways in **autoimmune**
:ase of the central nervous system
ıderson D.E.; Sharpe A.H.; Hafler D.A.
E. Anderson, Medical Microbiology and Immunology, University CA Davis
:hool Medicine, Tupper Hall, Davis, CA 95616 United States
)THOR EMAIL: deanderson@ucdavis.edu
ırrent Opinion in Immunology (CURR. OPIN. IMMUNOL.) (United Kingdom)
), 11/6 (677-683)
)DEN: COPIE ISSN: 0952-7915
)CUMENT TYPE: Journal; Review
\NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
)MBER OF REFERENCES: 36

'3/22 (Item 16 from file: 73)
)OG(R)File 73:EMBASE
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)1971 EMBASE No: 1999275111
lhesion and lymphocyte costimulatory molecules in systemic rheumatic
:ases
ikakis P.P.; Mavrikakis M.
. P.P. Sfikakis, 3 Amaryllidos Str., 154 52 Athens Greece
)THOR EMAIL: psfikaki@otenet.gr
inical Rheumatology (CLIN. RHEUMATOL.) (Belgium) 1999, 18/4
:17-327)
)DEN: CLRHD ISSN: 0770-3198
)CUMENT TYPE: Journal; Review
\NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
)MBER OF REFERENCES: 129

3/23 (Item 17 from file: 73)
)OG(R)File 73:EMBASE
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'3810 EMBASE No: 1999200181
'LA-4 and T cell activation
)sterwegel M.A.; Greenwald R.J.; Mandelbrot D.A.; Lorsbach R.B.; Sharpe
A. Oosterwegel, Immunology Research Division, Department of Pathology,
igham and Women's Hospital, 221 Longwood Avenue, Boston, MA 02115
ıited States
)THOR EMAIL: moosterwegel@rics.bwh.harvard.edu

irrent Opinion in Immunology (CURR. OPIN. IMMUNOL.) (United Kingdom)
, 11/3 (294-300)
DEN: COPIE ISSN: 0952-7915
CUMENT TYPE: Journal; Review
NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
MBER OF REFERENCES: 52

3/24 (Item 18 from file: 73)
OG(R)File 73:EMBASE
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8000 EMBASE No: 1999190730
cosal immunity and inflammation I. Mucosal dendritic cells: Their
ialized role in initiating T cell responses
asaki A.; Kelsall B.L.
L. Kelsall, National Institutes of Health, Bldg. 10, 10 Center Dr.,
thesda, MD 20892-1890 United States
THOR EMAIL: kelsall@nih.gov
erican Journal of Physiology - Gastrointestinal and Liver Physiology (
. J. PHYSIOL. GASTROINTEST. LIVER PHYSIOL.) (United States) 1999,
6/5 39-5 (G1074-G1078)
DEN: APGPD ISSN: 0193-1857
CUMENT TYPE: Journal; Review
NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
MBER OF REFERENCES: 24

3/25 (Item 19 from file: 73)
OG(R)File 73:EMBASE
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6926 EMBASE No: 1998327854
28/**B7** costimulation: A **review**
eenfield E.A.; Nguyen K.A.; Kuchroo V.K.
A. Greenfield, Department of Adult Oncology, Dana Farber Cancer
stitute, Boston, MA 02115 United States
itical Reviews in Immunology (CRIT. REV. IMMUNOL.) (United States)
98, 18/5 (389-418)
DEN: CCRID ISSN: 1040-8401
CUMENT TYPE: Journal; Review
NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
MBER OF REFERENCES: 229

3/26 (Item 20 from file: 73)
OG(R)File 73:EMBASE
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3604 EMBASE No: 1998320503
stimulatory molecles, CD 80 and CD 86
kasaki Y.
umachi (RYUMACHI) (Japan) 1998, 38/4 (623-630)
DEN: RYMCA ISSN: 0300-9157
CUMENT TYPE: Journal; Review
NGUAGE: JAPANESE
MBER OF REFERENCES: 45

3/27 (Item 21 from file: 73)
OG(R)File 73:EMBASE
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9384 EMBASE No: 1998199909
e cellular biology of B-cell chronic lymphocytic leukemia
rlander J.

Jurlander, Department of Hematology, Finsencenter, Rigshospitalet,
00 Copenhagen Denmark
itical Reviews in Oncology/Hematology (CRIT. REV. ONCOL. HEMATOL.) (
eland) 1998, 27/1 (29-52)
)DEN: CCRHE ISSN: 1040-8428
BLISHER ITEM IDENTIFIER: S1040842897100087
)CUMENT TYPE: Journal; Review
NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
MBER OF REFERENCES: 266

3/28 (Item 22 from file: 73)
OG(R)File 73:EMBASE
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0418 EMBASE No: 1998173817
cell activation in **autoimmune** and inflammatory diseases
rkins D.L.
. D.L. Perkins, Brigham and Women's Hospital, Lab. of
munogen./Transplantation, 75 Francis Street, Boston, MA 02115 United
ates
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)CUMENT TYPE: Journal; Article
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MBER OF REFERENCES: 56

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)CUMENT TYPE: Journal; Review
NGUAGE: ENGLISH
MBER OF REFERENCES: 545

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NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
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)G(R)File 73:EMBASE
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:rin P.J.; Racke M.K.
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:es Building, Philadelphia, PA 19104-4283 United States
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:UMENT TYPE: Journal; Review
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C 3631 EMBASE No: 1997010093
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erling A.I.; Bluestone J.A.
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iversity of Chicago, 5841 S Maryland Ave, Chicago, IL 60637 United
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NGUAGE: ENGLISH
MBER OF REFERENCES: 93

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C 5737 EMBASE No: 1997007199
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ı Herrath M.G.; Oldstone M.B.A.
3. Von Herrath, Scripps Research Institute, Department of
ıropharmacology, Division of Virology, 10666 North Torrey Pines Road,
Jolla, CA 92037 United States
THOR EMAIL: mbaobo@scripps.edu
:rent Opinion in Immunology (CURR. OPIN. IMMUNOL.) (United Kingdom)
J , 8/6 (878-885)
)EN: COPIE ISSN: 0952-7915
:UMENT TYPE: Journal; Review
NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
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vol E.A.; Schweitzer A.N.; Sharpe A.H.
A. Tivol, Blood Center Southeastern Wisconsin, Milwaukee, WI 53201-2178
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THOR EMAIL: btivol@smtpgate.bcsew.edu
:rent Opinion in Immunology (CURR. OPIN. IMMUNOL.) (United Kingdom)
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CUMENT TYPE: Journal; Conference Paper
NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

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ser H.; Stadecker M.J.
partment of Pathology, Tufts University School of Medicine, 136
rison Ave.,Boston, MA 02111 United States
v England Journal of Medicine (NEW ENGL. J. MED.) (United States)
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CUMENT TYPE: Journal; Review
NGUAGE: ENGLISH

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hcock K.S.; Hodes R.J.
perimental Immunology Branch, National Cancer Institute, National
stitutes of Health,Bethesda, MD 20892 United States
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-mediated costimulation and the immune response
ıultze J.; Nadler L.M.; Gribben J.G.
/ision of Hematologic Malignancies, Dana Farber Cancer Institute,
)artment of Medicine,Boston, MA 02115 United States
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)EN: BLORE ISSN: 0268-960X
:UMENT TYPE: Journal; Review
NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

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D)G(R)File 73:EMBASE
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ıschow D.J.; Walunas T.L.; Bluestone J.A.
)artment of Pathology, Ben May Institute, University of
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ıual Review of Immunology (ANNU. REV. IMMUNOL.) (United States) 1996
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NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

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)mas R.; Lipsky P.E.
)artment of Medicine, University of Queensland, Princess Alexandra
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:hritis and Rheumatism (ARTHRITIS RHEUM.) (United States) 1996, 39/2
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:UMENT TYPE: Journal; Review
NGUAGE: ENGLISH

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:rin P.J.; Scott D.; June C.H.; Racke M.K.
nune Cell Biology Program, Naval Medical Research Institute, Mail Stop
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Listol-Myers Squibb Pharm Res Inst, 3005 First Avenue,Seattle, WA 98121
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CUMENT TYPE: Journal; Review
NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

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iestone J.A.
i May Institute, Department of Pathology, University of
icago,Chicago, IL 60637 United States
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)EN: IUNIE ISSN: 1074-7613
CUMENT TYPE: Journal; Review
NGUAGE: ENGLISH

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(2001 Elsevier Science B.V. All rts. reserv.

0(7198 EMBASE No: 1995193236
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tman A.P.
)t. Medicine, University Sheffield, Clinical Sciences Centre, Northern
ieral Hospital,Sheffield S5 7AU United Kingdom
irnal of Autoimmunity (J. AUTOIMMUN.) (United Kingdom) 1995, 8/3
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)EN: JOAUE ISSN: 0896-8411
CUMENT TYPE: Journal; Review
NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

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L Carpus W.J.
)artment Microbiology-Immunology, Northwestern Univ. Medical School,
E Chicago Avenue,Chicago, IL 60611 United States
munological Reviews (IMMUNOL. REV.) (Denmark) 1995, -/144 (225-244)
)EN: IMRED ISSN: 0105-2896
CUMENT TYPE: Journal; Review
NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

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lovnick A.D.; Ebers G.C.
)artment of Medical Genetics, University of British Columbia, 222-6174
Iversity Boulevard,Vancouver, BC V6T 1Z3 Canada
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:UMENT TYPE: Journal; Review
NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

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.gle W.O.
)artment of Immunology, Scripps Research Institute,San Diego, CA
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:UMENT TYPE: Journal; Review
NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

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/ision of Hematologic Oncology, Memorial Sloan-Kettering Cancer Ctr.,
402, 1275 York Avenue,New York, NY 10021 United States
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)EN: CUOOE ISSN: 1040-8746
:UMENT TYPE: Journal; Review
NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

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/ision of Hematologic Malignancies, Dana-Farber Cancer Institute, 44
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NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

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0 1415 EMBASE No: 1994047566
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:tion of Immunobiology, Howard Hughes Medical Institute, Yale
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:UMENT TYPE: Journal; Review
NGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

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0 5707 EMBASE No: 1989175703
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vsom-Davis J.
)artment of Clinical Neurology, Radcliffe Infirmary, University of
ford, Oxford OX2 6HE United Kingdom
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:UMENT TYPE: Journal
NGUAGE: ENGLISH

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:tion of Immunobiology, Yale University School of Medicine, New Haven,
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nguages: ENGLISH
:ument type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

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:ranyi GG; Benczur M; Varga M; Gyorffy G; Gyodi E; Onody K; Hollan RS
ıales immunologiae Hungaricae (HUNGARY) 1979, 19 p93-104, ISSN
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3/56 (Item 1 from file: 399)
D)G(R)File 399:CA SEARCH(R)
(2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

1241571 CA: 131(18)241571r CONFERENCE PROCEEDING
rgeting costimulatory and other signaling molecules in murine lupus
'HOR(S): Halvorson, Mark J.; Gause, William C.
:ATION: Department of Microbiology and Immunology, Uniformed Services
Ui ersity of Health Sciences, Bethesda, MD, USA
JRNAL: Lupus EDITOR: Kammer, Gary M. (Ed), Tsokos, George C (Ed),
'E: 1999 PAGES: 656-670 CODEN: 67VNAV LANGUAGE: English PUBLISHER:
H ıa,Totowa, N. J

3/57 (Item 2 from file: 399)
D)G(R)File 399:CA SEARCH(R)
(2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

1003885 CA: 131(1)3885b JOURNAL
ıunomodulation of the CD28-B7 system: effects of inhibition of
c :imulatory signals provided by CD28-B7 interaction on rat autoimmune
a -glomerular basement membrane glomerulonephritis
'HOR(S): Nishikawa, K.; Matsuo, S.
:ATION: Division of Nephrology, The Third Department of Internal
M. :ine, Nagoya University School of Medicine, Nagoya, Japan, 466-8550
JRNAL: Nephrol., Dial., Transplant. DATE: 1999 VOLUME: 14 NUMBER:
S 1. 1 PAGES: 19-21 CODEN: NDTREA ISSN: 0931-0509 LANGUAGE: English
BLISHER: Oxford University Press

3/58 (Item 3 from file: 399)
D)G(R)File 399:CA SEARCH(R)
(2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

3009996 CA: 128(2)9996y JOURNAL
-immunotoxin for in vivo modulation of primary immune responses
'HOR(S): Otten, Henny G.; De Gast, Gijsbert C.
:ATION: Dept. of Medical Immunology, Transplantation Laboratory,
U ersity Hospital Utrecht, 3584 CX, Utrecht, Neth.
JRNAL: Drug News Perspect. DATE: 1997 VOLUME: 10 NUMBER: 7 PAGES:
4 105 CODEN: DNPEED ISSN: 0214-0934 LANGUAGE: English PUBLISHER:
P ;

3/59 (Item 4 from file: 399)
D)G(R)File 399:CA SEARCH(R)
(2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

/344987 CA: 127(25)344987c CONFERENCE PROCEEDING
: yin/yang of CD28 T-cell costimulation in autoimmunity
'HOR(S): Bluestone, Jeffrey A.; Miller, Stephen
:ATION: Dep. of Pathology and Committee on Immunology, Ben May
I: ıtute for Cancer Research, University of Chicago, Chicago, IL, 60637,
U:
JRNAL: Immune Tolerance, Int. Symp. EDITOR: Banchereau, Jacques (Ed),
'E: 1996 PAGES: 105-113 CODEN: 65FCA6 LANGUAGE: English PUBLISHER:
E /ier,Paris, Fr

3/60 (Item 5 from file: 399)
DG(R)File 399:CA SEARCH(R)
(c 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

7219098 CA: 127(16)219098n JOURNAL
v strategy for immune regulation
THOR(S): Okumura, Ko; Azuma, Miyuki
ATION: Menekigaku, Juntendo Daigaku, Tokyo, Japan, 113
URNAL: Nippon Naika Gakkai Zasshi DATE: 1997 VOLUME: 86 NUMBER: 9
ES: 1778-1783 CODEN: NNGAAS ISSN: 0021-5384 LANGUAGE: Japanese
BLISHER: Nippon Naika Gakkai

3/61 (Item 6 from file: 399)
DG(R)File 399:CA SEARCH(R)
(c 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

5210644 CA: 126(16)210644j JOURNAL
28/B7 regulation of autoimmune diabetes
THOR(S): Herold, Kevan C.; Lenschow, Deborah J.; Bluestone, Jeffrey A.
ATION: Department of Medicine, The University of Illinois at Chicago,
C igo, IL, 60612, USA
URNAL: Immunol. Res. DATE: 1997 VOLUME: 16 NUMBER: 1 PAGES: 71-84
DEN: IMRSEB ISSN: 0257-277X LANGUAGE: English PUBLISHER: Humana

3/62 (Item 7 from file: 399)
DG(R)File 399:CA SEARCH(R)
(c 2001 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

3007401 CA: 123(1)7401y JOURNAL
e CD28/CTLA-4:B7 receptor system in experimental autoimmune
e phalomyelitis
THOR(S): Linsley, Peter S.
ATION: Bristol -Myers Squibb Pharmaceutical Res. Inst., Seattle, WA,
U
URNAL: J. Clin. Invest. DATE: 1995 VOLUME: 95 NUMBER: 6 PAGES:
2 -30 CODEN: JCINAO ISSN: 0021-9738 LANGUAGE: English

ASE ENTER A COMMAND OR BE LOGGED OFF IN 5 MINUTES
? s (b7 or b7(w)1) and autoimmun?

```
          13268  B7
          13268  B7
        7503937  1
           3860  B7(W)1
         167227  AUTOIMMUN?
     S7     966  (B7 OR B7(W)1) AND AUTOIMMUN?
? s s7 and (sclerosis or diabetes)

            966  S7
         139604  SCLEROSIS
         444184  DIABETES
     S8     264  S7 AND (SCLEROSIS OR DIABETES)
? s s8 and review?

            264  S8
        2851120  REVIEW?
     S9      21  S8 AND REVIEW?
? rd s9

...completed examining records
    S10      15  RD S9 (unique items)
? t s10/7/all
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10/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12181396 BIOSIS NO.: 199900476245
Differential requirements of naive and memory T cells for CD28 costimulation in **autoimmune** pathogenesis.
AUTHOR: Perrin P J(a); Lovett-Racke A; Phillips S M; Racke M K
AUTHOR ADDRESS: (a)Department of Medicine, University of Pennsylvania, 909 BRB II, 421 Curie Boulevard, Philadelphia, PA, 19104-6160**USA
JOURNAL: Histology and Histopathology 14 (4):p1269-1276 Oct., 1999
ISSN: 0213-3911
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Experimental **autoimmune** encephalomyelitis (EAE) is the most extensively studied animal model of the human disease multiple **sclerosis** (MS). In EAE, CNS demyelination is induced by immunization with myelin proteins or adoptive transfer of myelin-reactive CD4+ T cells. Since the antigen specificity of the immune response believed to be responsible for the pathology of MS is not well defined, therapies that target aspects of T cell activation that are not antigen specific may be more applicable to the treatment of MS. As a result, understanding the role of costimulatory molecules in the activation of naive and memory T cells has become an area of extensive investigation. Naive T cells require two signals for activation. Signal one is provided by engagement of the T cell receptor (TCR) with MHC/peptide complexes and provides antigen specificity to the immune response. The second signal, termed costimulation, is usually provided by **B7** molecules on APC to CD28 molecules expressed on T cellsand is antigen-independent. This **review** will discuss our current understanding of costimulation in

the induction and perpetuation of EAE, as well as the potential of costimulaton blockade in the treatment of MS.

10/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12150299 BIOSIS NO.: 199900445148
Multiple **sclerosis** : Pathology and pathogenesis.
AUTHOR: Prasad Rameshwar(a); Prasad Roli(a); Wacek Bartholomew(a); Chandran Rajeswari(a); Ilangovan Saroja(a)
AUTHOR ADDRESS: (a)Department of Pathology, University of Illinois at Chicago, 1819 West Polk Street, Chicago, IL, 60612-7335**USA
JOURNAL: Proceedings of the National Academy of Sciences India Section B (Biological Sciences) 68 (3-4):p189-198 1998
ISSN: 0369-8211
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Multiple **Sclerosis** (MS) is a disease that results in the demyelination of axons in the central nervous system. The etiology of this disease is not completely known. The environment, genetics, viruses, and/or the immune system are considered to be factors in the progression of this debilitating disease. However, the exact combination of these factors that leads to the pathogenesis of MS is not known. It is known that MS is more prevalent in the northern hemisphere, especially in the United Kingdom's Orkney and Shetland Islands and in the northern United States of America. In India, MS is prevalent in the Zoroastrian population. This disease is also more prevalent in women, whites, and individuals with a family hisotry of multiple **sclerosis**. Intrathecal secretion of IgG and the appearance of oligoclonal bands on agar-gel electrophoresis indicate an immunological etiology of MS. If the etiology is immunological, the immune response might be triggered by any one or combination of the previously mentioned factors (i.e., environmental, genetic, viral, etc.). Studies show that the humoral immune response in MS is modulated by histocompatibility antigens. An **autoimmune** hypothesis for this disease stems from a strong association with HLA antigens, mainly HLA-3, HLA- **B7**, and HLA-DR2. To date, no specific test is available which can conclusively diagnose MS. Increased concentration of cerebral spinal fluid (CSF) IgG, presence of oligoclonal bands on protein gel electrophoresis, and increased concentration of myelin basic protein (MBP), are indicative of this disease. Magnetic Resonance Imaging (MRI) scanning is a recent diagnostic tool which clearly shows the size, quantity, and distribution of lesions or plaques. MRI results, in combination with medical history, neurological examination, and laboratory information, can significantly help in the diagnosis of MS. This **review** focuses on the pathological features and pathogenetic mechanisms in multiple **sclerosis**.

10/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11669616 BIOSIS NO.: 199800451347
Targeting the **B7** /CD28: CTLA-4 costimulatory system in CNS **autoimmune** disease.
AUTHOR: Karandikar Nitin J; Vanderlugt Carol L; Bluestone Jeffrey A; Miller Stephen D(a)
AUTHOR ADDRESS: (a)Dep. Microbiol.-Immunol. Interdepartmental Immunobiol. Cent., North Western Univ. Med. Sch., 303**USA
JOURNAL: Journal of Neuroimmunology 89 (1-2):p10-18 Aug. 14, 1998

ISSN: 0165-5728
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The **B7**/CD28:CTLA-4 costimulatory pathway plays a critical role in determining the fate of immune responses (activation vs. down-regulation) and is a highly promising therapeutic target for treating **autoimmune** diseases. In this **review**, we highlight the mechanisms by which this costimulatory pathway operates emphasizing the role of the different components in the pathogenesis of relapsing experimental **autoimmune** encephalomyelitis, a CD4 T cell-mediated **autoimmune** model of multiple **sclerosis**. The separate and distinct roles of **B7-1**, **B7-2** and CTLA-4 in positive and negative regulation of **autoimmune** pathogenesis are considered and a working model is proposed.

10/7/4 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

10927362 EMBASE No: 1998331678
T-cell anergy and altered t-cell receptor signaling: Effects on **autoimmune** disease
Salojin K.V.; Zhang J.; Madrenas J.; Delovitch T.L.
K.V. Salojin, Autoimmunity/Diabetes Group, Dept of Microbiology, University of Western Ontario, London, Ont. N6A 5K8 Canada
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Immunology Today (IMMUNOL. TODAY) (United Kingdom) 1998, 19/10 (468-473)
CODEN: IMTOD ISSN: 0167-5699
PUBLISHER ITEM IDENTIFIER: S0167569998013267
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 71

Immunological self-tolerance can be acquired by several mechanisms, including the induction of anergy in autoreactive T cells. In this sense, anergy is predictably advantageous for the immune system. Here, Konstantin Salojin and colleagues present an alternative view that the induction of anergy in regulatory T cells may be harmful to the host and elicit **autoimmune** disease.

10/7/5 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

10561079 EMBASE No: 2000025912
Molecular pathogenesis of multiple **sclerosis**
Bar-Or A.; Oliveira E.M.L.; Anderson D.E.; Hafler D.A.
D.A. Hafler, Center for Neurologic Diseases, Brigham and Women's Hospital, Harvard Medical School, 77 Avenue Louis Pasteur, Boston, MA 02115-5187 United States
AUTHOR EMAIL: hafler@cnd.bwh.harvard.edu
Journal of Neuroimmunology (J. NEUROIMMUNOL.) (Netherlands) 1999, 100/1-2 (252-259)
CODEN: JNRID ISSN: 0165-5728
PUBLISHER ITEM IDENTIFIER: S0165572899001939
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 96

Multiple **sclerosis** (MS) is best understood as an inflammatory disease of the central nervous system (CNS) white matter characterized by

demyelination, focal T cell and macrophage infiltrates, axonal injury and loss of neurological function. Our current understanding invokes proinflammatory cells and mediators that may be triggered by environmental factors to mediate disease in a genetically susceptible host. Five major themes which have been associated with the pathogenesis of MS lesions will be discussed: (1) The differential activation states of myelin-reactive T cells from MS patients vs. normal individuals, (2) the selective expression of chemokines, adhesion molecules and matrix metalloproteinases, (3) the proposed roles of the **B7** costimulatory pathway, (4) the proinflammatory cytokines and (5) the role of molecular mimicry.

10/7/6 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

07791971 EMBASE No: 1999275111
Adhesion and lymphocyte costimulatory molecules in systemic rheumatic diseases
Sfikakis P.P.; Mavrikakis M.
Dr. P.P. Sfikakis, 3 Amaryllidos Str., 154 52 Athens Greece
AUTHOR EMAIL: psfikaki@otenet.gr
Clinical Rheumatology (CLIN. RHEUMATOL.) (Belgium) 1999, 18/4 (317-327)
CODEN: CLRHD ISSN: 0770-3198
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 129

Adhesion molecules expressed on the surface of immune cells transduce a variety of cell-activating signals and mediate important interactions by binding to multiple specific counter-receptors expressed on other cells or on extracellular matrix components. A large number of aberrations in the expression of cell-bound molecules at the mRNA and protein level in vivo have been described in patients with **autoimmune** connective tissue diseases. In vitro studies suggest the presence of functional abnormalities of adhesive pathways, at least at some points of the disease. Increased circulating levels of isoforms of several adhesion molecules have also been demonstrated in these patients. The possible involvement in the pathogenesis of rheumatoid arthritis, systemic lupus erythematosus, Sjogren's syndrome and systemic **sclerosis** of E-, P- and L-selectins, of some integrins and of several adhesion molecules of the immunoglobulin superfamily that in addition participate in lymphocyte costimulation will be discussed in this **review**. Further studies on migration and recruitment patterns of immune cells into inflamed tissues, as well as on possible defects of lymphocyte activation in these patients, are expected to expand our knowledge on systemic **autoimmune** responses and identify targets for specific immunotherapy.

10/7/7 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

07436926 EMBASE No: 1998327854
CD28/**B7** costimulation: A **review**
Greenfield E.A.; Nguyen K.A.; Kuchroo V.K.
E.A. Greenfield, Department of Adult Oncology, Dana Farber Cancer Institute, Boston, MA 02115 United States
Critical Reviews in Immunology (CRIT. REV. IMMUNOL.) (United States) 1998, 18/5 (389-418)
CODEN: CCRID ISSN: 1040-8401
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 229

The current model of T cell activation requires two signals. The first signal is specific, requiring T cell receptor recognition and binding to MHC/Antigen presented by an antigen-presenting cell. The second signal is nonspecific, resulting from the binding of **B7** ligand on the antigen-presenting cell with its receptor, CD28, on the T cell. If both signals are provided, the T cell will proliferate and secrete cytokines. Recently, it has been shown that CTLA4, another receptor for **B7** that is upregulated following T cell after activation, can deliver an inhibitory signal, downregulating T cell proliferation. The **B7** family of ligands has two family members, **B7-1** and **B7**-2. They both bind to CD28 and CTLA4, but they differ in their binding affinity, structure, and temporal expression. Considerable research has been done on the CD28/**B7** costimulatory pathway. Different ways of manipulating this pathway could provide insights into the mechanism and treatment of opposing pathological states. Blocking the CD28/**B7** pathway could result in immunosuppression, with implications for the treatment of **autoimmune** diseases, organ transplantation, and graft vs. host disease. Activating the CD28/**B7** pathway could be useful for including the immune system to recognize and eliminate tumors that evade the immune system. Finally, the CD28/**B7** pathway could be involved with maintaining immune to tolerance, as recent studies suggest the preferential binding of the **B7**-CTLA4 pathway results in the down-regulation of the responding T cells. Thus, the **B7**/CD28/CTLA4 pathway has the ability to both positively and negatively regulate immune responses.

10/7/8 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

06994545 EMBASE No: 1997280717
Targeting **B7**: CD28 Cco-stimulation in the tratment of **autoimmune** demyelination
Perrin P.J.; Racke M.K.
Dr. P.J. Perrin, Dept, of Neurology, University of Pennsylvania, 3 W. Gates Building, Philadelphia, PA 19104-4283 United States
Drug News and Perspectives (DRUG NEWS PERSPECT.) (Spain) 1997, 10/4 (208-213)
CODEN: DNPEE ISSN: 0214-0934
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 56

10/7/9 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

06725737 EMBASE No: 1997007199
Virus-induced **autoimmune** disease
Von Herrath M.G.; Oldstone M.B.A.
M.G. Von Herrath, Scripps Research Institute, Department of Neuropharmacology, Division of Virology, 10666 North Torrey Pines Road, La Jolla, CA 92037 United States
AUTHOR EMAIL: mbaobo@scripps.edu
Current Opinion in Immunology (CURR. OPIN. IMMUNOL.) (United Kingdom) 1996, 8/6 (878-885)
CODEN: COPIE ISSN: 0952-7915
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 73

The breaking of tolerance or unresponsiveness to self-antigens, involving the activation of autoreactive lymphocytes, is a critical event leading to **autoimmune** diseases. The precise mechanisms by which this can occur

are mostly unknown. Viruses have been implicated in this process, among other etiological factors, such as genetic predisposition and cytokine activity. Several ways have been proposed by which a viral infection might break tolerance to self and trigger an autoreactive cascade that ultimately leads to the destruction of a specific cell type or an entire organ. The process termed 'molecular mimicry' and the use of transgenic models in which viral and host genes can be manipulated to analyze their effects in causing **autoimmunity** have been particular focuses for research. For example, there is a transgenic murine model of virus-induced **autoimmune** disease, in which a known viral gene is selectively expressed as a self-antigen in beta cells of the pancreas. In these mice, insulin-dependent **diabetes** develops after either a viral infection, the release of a cytokine such as IFN-gamma, or the expression of the costimulatory molecule **B7.1** in the islets of Langerhans. Recent studies using this model have contributed to the understanding of the pathogenesis of virus-induced **autoimmune** disease and have furthered the design and testing of novel immunotherapeutic approaches.

10/7/10 (Item 7 from file: 73)
DIALOG(R)File 73:EMBASE

06551821 EMBASE No: 1996212396
B7-mediated costimulation and the immune response
Schultze J.; Nadler L.M.; Gribben J.G.
Division of Hematologic Malignancies, Dana Farber Cancer Institute, Department of Medicine,Boston, MA 02115 United States
Blood Reviews (BLOOD REV.) (United Kingdom) 1996, 10/2 (111-127)
CODEN: BLORE ISSN: 0268-960X
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

In addition to presentation of antigen, T cells require additional or 'costimulatory' signals from antigen-presenting cells. Failure to receive costimulation following antigen presentation renders T cells anergic, and these cells are functionally incapable of proliferating or secreting cytokines in response to subsequent rechallenge. Recent evidence has demonstrated that a critical costimulatory signal is delivered by members of the **B7** family. **B7-1** (CD80) and **B7**-2 (CD86) provide costimulation through CD28, their ligand on the T cell. Dysregulation of expression of **B7** may be implicated in the pathogenesis of **autoimmune** disease. In contrast, lack of expression of **B7** on tumor cells may explain in part the lack of immune response against the majority of tumors. It may now be possible to exploit this pathway to induce immunological response against tumors. Blockade of this pathway will likely have significant impact on transplantation biology, to induce T-cell anergy and prevent graft rejection and graft-versus-host disease.

10/7/11 (Item 8 from file: 73)
DIALOG(R)File 73:EMBASE

06132471 EMBASE No: 1995164038
Evolution of the T-cell repertoire during the course of experimental immune-mediated demyelinating diseases
Miller S.D.; McRae B.L.; Vanderlugt C.L.; Nikcevich K.M.; Pope J.G.; Pope L.; Karpus W.J.
Department Microbiology-Immunology, Northwestern Univ. Medical School, 303 E Chicago Avenue,Chicago, IL 60611 United States
Immunological Reviews (IMMUNOL. REV.) (Denmark) 1995, -/144 (225-244)
CODEN: IMRED ISSN: 0105-2896
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

Fig. 6 depicts a model for epitope spreading in T cell-mediated demyelination. The acute phase of disease is due to T cells specific for the initiating epitope, which can be either a determinant on the CNS target organ of the **autoimmune** response or a determinant on a persisting, CNS-tropic virus. The primary T cell response is responsible for the initial tissue damage by the production of pro-inflammatory Th1 cytokines which can affect myelination directly and indirectly by their ability to recruit and activate macrophages to phagocytize myelin. As a result of myelin damage and opening of the blood-brain-barrier during acute disease, T cells specific for endogenous epitopes on the same and/or different myelin proteins are primed and expand either in the periphery or locally in the CNS. These secondary T cells initiate an additional round of myelin destruction, leading to a clinical relapse by production of additional pro-inflammatory cytokines, similar to the bystander demyelination operative during acute disease. It will be of great interest to determine the relative contributions of local and systemic immune responses to these endogenous neuroepitopes. It is possible that local CNS presentation of endogenous neuroepitopes following acute CNS damage could be mediated by infiltrating inflammatory macrophages, activated microglial cells, endothelial cells and/or astrocytes. These tissue resident antigen presenting cells have been shown to upregulate expression of MHC class II, certain adhesion molecules, and **B7** costimulatory molecules in response to pro-inflammatory cytokines. The data on epitope spreading provided by the murine demyelinating disease models clearly illustrate the dynamic nature of the T cell repertoire during chronic inflammation in a specific target organ. The contribution of epitope spreading to chronic CNS demyelination could be considered to be a special case since tolerance to myelin epitopes would be expected to be inefficient due to their sequestration behind the blood-brain-barrier. However, the recent description of epitope spreading in response to pancreatic antigens in spontaneous **diabetes** in the NOD mouse may indicate that this phenomenon is operative in a variety of organ-specific experimental and spontaneous **autoimmune** diseases. Thus, potential therapy for the advanced stages of relapsing EAE, and perhaps other organ-specific experimental and human **autoimmune** diseases exhibiting chronic/relapsing-remitting clinical courses, would need to be directed not only against the effector cells initiating the disease, but also against effector cells With novel specificities (which likely utilize different TCR alpha and beta chains) recruited as a result of tissue damage.

06032252 EMBASE No: 1995062490
Genetics of multiple **sclerosis**
Sadovnick A.D.; Ebers G.C.
Department of Medical Genetics, University of British Columbia, 222-6174 University Boulevard,Vancouver, BC V6T 1Z3 Canada
Neurologic Clinics (NEUROL. CLIN.) (United States) 1995, 13/1 (99-118)
CODEN: NECLE ISSN: 0733-8619
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

The cause of MS is unknown. There is considerable circumstantial evidence that MS is a complex trait, probably **autoimmune** in nature, and is determined by both genetic and environmental factors. At present, it must be acknowledged, however, that our understanding of the pathogenesis of MS is minimal. Very little is known about the genes determining disease susceptibility and perhaps even less is understood about environmental factors that influence penetrance or the geographic distribution. This lack of knowledge results neither from lack of effort nor from a shortage of fertile imaginations. Almost every imaginable hypothesis has, in the past, found some support. The intractability of the problem could well result

from its complexity, because answers to testable hypotheses are commonly negative or ambiguous. Today, the opportunity exists for researchers to provide such answers because of recent major developments. The first development is the recognition that MS research requires a relatively large pool of well- ascertained, carefully diagnosed, and longitudinally well-characterized MS patients. The last two developments are the identification and successful application of statistical and molecular genetic techniques carrying sufficient power to allow the exploration of complex traits such as MS.

10/7/13 (Item 10 from file: 73)
DIALOG(R)File 73:EMBASE

00856123 EMBASE No: 1977201740
HLA antigen segregation analysis in multiple **sclerosis** (MS) families
Bertrams J.; Kuwert E.
Abt. Lab. Med., Elisabeth Krankenh., Essen Germany
Zeitschrift fur Immunitatsforschung (Z. IMMUNITATSFORSCH.) 1976, 152/3 (200-208)
CODEN: ZIEKB
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH

HLA antigen segregation analysis was performed in 38 families with a total of 52 multiple **sclerosis** (MS) patients including 14 families with 2 affected sibs. HLA A3 and especially **B7** occurred more frequently in familial MS cases (propositi) (A3: 42.9%, **B7**: 64.3%) than in non familial cases (A3: 35.8%, **B7**: 35.3%). As could be expected from the increased phenotype frequencies of A3 and **B7**, the haplotype A3-**B7** showed a disturbed segregation among the MS patients but not among their healthy sibs. A second haplotype, A1-B8, was found more frequently than expected among the MS patients but also among their unaffected sibs. These data may suggest increased MS susceptibility associated with the haplotype A3-**B7** and, conversely **autoimmune** or protective properties associated with A1-B8 in MS patients and their healthy sibs, respectively.

10/7/14 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

126210644 CA: 126(16)210644j JOURNAL
CD28/B7 regulation of autoimmune diabetes
AUTHOR(S): Herold, Kevan C.; Lenschow, Deborah J.; Bluestone, Jeffrey A.
LOCATION: Department of Medicine, The University of Illinois at Chicago, Chicago, IL, 60612, USA
JOURNAL: Immunol. Res. DATE: 1997 VOLUME: 16 NUMBER: 1 PAGES: 71-84
CODEN: IMRSEB ISSN: 0257-277X LANGUAGE: English PUBLISHER: Humana
SECTION:
CA215000 Immunochemistry
IDENTIFIERS: review CD28 B7 antigen autoimmune diabetes
DESCRIPTORS:
CD28(antigen)... CD80(antigen)... Immune tolerance... Insulin-dependent diabetes mellitus...
CD28/B7 regulation of autoimmune diabetes
CAS REGISTRY NUMBERS:
18883-66-4 autoimmune diabetes induced by; CD28/B7 regulation of autoimmune diabetes

10/7/15 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123007401 CA: 123(1)7401y JOURNAL

The CD28/CTLA-4:B7 receptor system in experimental autoimmune encephalomyelitis

AUTHOR(S): Linsley, Peter S.

LOCATION: Bristol -Myers Squibb Pharmaceutical Res. Inst., Seattle, WA, USA

JOURNAL: J. Clin. Invest. DATE: 1995 VOLUME: 95 NUMBER: 6 PAGES: 2429-30 CODEN: JCINAO ISSN: 0021-9738 LANGUAGE: English

SECTION:
CA215000 Immunochemistry

IDENTIFIERS: CD28 CTLA4 B7 autoimmune encephalomyelitis review

DESCRIPTORS:
Antigens,B7/BB-1... Antigens,CD28... Antigens,CTLA-4 (cytotoxic T-lymphocyte-activating, 4)... Encephalomyelitis,autoimmune... Lymphocyte,B-cell... Lymphocyte,T-cell... Multiple sclerosis...

CD28/CTLA-4:B7 receptor system in exptl. autoimmune encephalomyelitis

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Set     Items   Description
S1          6   AU="ANDERSON DARRELL R"
S2          6   RD S1 (unique items)
S3          6   (16C10 OR 7C10 OR 20C9 OR 7B6) AND (B7 OR B7(W)1)
S4          2   RD S3 (unique items)
S5         90   (B7 OR B7(W)1) AND PSORIASIS
S6         51   RD S5 (unique items)
S7        966   (B7 OR B7(W)1) AND AUTOIMMUN?
S8        264   S7 AND (SCLEROSIS OR DIABETES)
S9         21   S8 AND REVIEW?
S10        15   RD S9 (unique items)
? s s7 and review?

            966  S7
        2851120  REVIEW?
    S11      84  S7 AND REVIEW?
? rd s11

...examined 50 records  (50)
...completed examining records
    S12      62  RD S11 (unique items)
? ds


Set     Items   Description
S1          6   AU="ANDERSON DARRELL R"
S2          6   RD S1 (unique items)
S3          6   (16C10 OR 7C10 OR 20C9 OR 7B6) AND (B7 OR B7(W)1)
S4          2   RD S3 (unique items)
S5         90   (B7 OR B7(W)1) AND PSORIASIS
S6         51   RD S5 (unique items)
S7        966   (B7 OR B7(W)1) AND AUTOIMMUN?
S8        264   S7 AND (SCLEROSIS OR DIABETES)
S9         21   S8 AND REVIEW?
S10        15   RD S9 (unique items)
S11        84   S7 AND REVIEW?
S12        62   RD S11 (unique items)
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60851

Functional CD86 (**B7**-2/B70) is predominantly expressed on Langerhans cells in atopic dermatitis [see comments]

Ohki O; Yokozeki H; Katayama I; Umeda T; Azuma M; Okumura K; Nishioka K

Department of Dermatology, Tokyo Medical and Dental University School of Medicine, Japan.

British journal of dermatology (ENGLAND) Jun 1997, 136 (6) p838-45, ISSN 0007-0963 Journal Code: AW0

Comment in Br J Dermatol 1998 Feb;138(2):358-9

Languages: ENGLISH

Document type: JOURNAL ARTICLE

Recently, we reported the functional expression of CD86 on cultured human Langerhans cells derived from normal epidermis. In the present study, we investigated the expression and function of co-stimulatory molecules in the pathogenesis of atopic dermatitis. In immunohistochemical analysis, CD80 and/or CD86 were detected on dendritic-shaped cells not only in the epidermis but also in the dermis in the inflammatory lesions of atopic dermatitis (n = 12). CD80 was expressed in only five cases (42%), while CD86 was expressed in all cases (100%). These molecules were not detected in normal control subjects (n = 8). In non-lesional skin of atopic dermatitis (n = 4), CD86 but not CD80 was detected in one case. CD86 was preferentially induced on dendritic-shaped cells in positive patch test sites to Dermatophagoides pteronyssinus or house dust allergen in atopic dermatitis (n = 4). The CD80- or CD86-positive cells were confirmed as Langerhans cells by double immunostaining using anti-CD1a monoclonal antibody. Neither CD86 nor CD80 was detected on keratinocytes. Similar results of the stronger expression of CD86 over that of CD80 were obtained from **psoriasis** vulgaris (n = 11) and from contact dermatitis (n = 7), although CD86 was expressed only in 57% of the contact dermatitis cases. The percentage of Langerhans cells positive for CD86 was higher than for CD80, i.e. 48% compared with 9%, respectively, in the epidermis of lesional skin of atopic dermatitis (n = 8). The expression rate of these molecules on Langerhans cells increased in the dermis. To investigate the function of co-stimulatory molecules on Langerhans cells in atopic dermatitis, we conducted an inhibition test with antibodies. Anti-CD86 monoclonal antibody almost completely inhibited T-cell proliferation stimulated with crude extract of D. pteronyssinus in the presence of epidermal cells as antigen-presenting cells, whereas anti-CD80 monoclonal antibody produced less of an inhibitory effect. These data indicate that CD86 expressed on Langerhans cells may play an important part in the pathogenesis of atopic dermatitis.

274686 EMBASE No: 1998158850

Enhanced expression of **B7**.2 (CD86) in patients with atopic dermatitis: A potential role in the modulation of Ige synthesis

Jirapongsananuruk O.; Hofer M.F.; Trumble A.E.; Norris D.A.; Leung D.Y.M.
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Journal of Immunology (J. IMMUNOL.) (United States) 01 MAY 1998, 160/9 (4622-4627)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 49

Recent studies have suggested that the accessory molecules **B7**.**1** (CD80) and **B7**.2 (CD86) differ in their capacity to generate Th1 vs Th2 responses. Atopic dermatitis (AD) is a chronic allergic skin disease associated with increased IgE synthesis. To determine the potential role of **B7**.2 molecules in AD, the present study was conducted to compare the expression of **B7**.**1** vs **B7**.2 on B cells from patients with AD vs normal subjects or patients with **psoriasis**. The expression of **B7**.2 on B cells of AD patients (53.67 +/- 3.10%) was significantly higher than normals (38.02 +/- 4.95%; p = 0.02) and **psoriasis** patients (40.19 +/- 2.70%; p = 0.006). In contrast, there was no significant difference in **B7**.**1** expression among the three subject groups. Interestingly, total serum IgE from AD patients and normal subjects correlated significantly with **B7**.2 expression on B cells (r = 0.68; p = 0.004), suggesting a role for **B7**.2sup + B cells in IgE synthesis. Indeed, purified **B7**.2sup + B cells produced significantly more IgE than **B7**.2- B cells in vitro (p = 0.04). Anti-human **B7**.2, but not **B7**.**1**, mAb significantly (p < 0.05) decreased IgE production by PBMC stimulated with IL-4 and anti-CD40 mAb. Furthermore, **B7**.2sup + B cells had a significantly higher level of IL-4R and CD23 expression than **B7**.1sup + B cells. These data demonstrate the predominant expression of **B7**.2 in AD, but not **psoriasis**, and a novel role for this molecule in IgE synthesis.

07643763 EMBASE No: 1999131477
Animal models of **psoriasis** - What can we learn from them?
Schon M.P.
Dr. M.P. Schon, Department of Dermatology, Heinrich-Heine-University, Moorenstr. 5, D-40225 Dusseldorf Germany
Journal of Investigative Dermatology (J. INVEST. DERMATOL.) (United States) 1999, 112/4 (405-410)
CODEN: JIDEA ISSN: 0022-202X
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 60

Research into the pathogenesis of **psoriasis** has been hampered by the lack of an animal disease resembling this common human skin disorder. Over the past few years, however, various rodent models that mirror aspects of the psoriatic phenotype and pathogenesis have become available. Here, the most prominent models are compared with human **psoriasis** and potential uses for **psoriasis** research are reviewed. Asebia (ab), flaky skin (fsn), and chronic proliferative dermatitis (cpd) are spontaneous mouse mutations with psoriasiform skin alterations of unclear pathogenesis. Transgenic mice with cutaneous overexpression of cytokines, such as interferon-gamma, interleukin- 11alpha, keratinocyte growth factor, transforming growth factor-alpha, interferon- 6, vascular endothelial growth factor, or bone morphogenic protein-6, are valuable tools for studying in vivo effects of individual cytokines in the pathogenesis of psoriasiform features. Psoriasiform lesions also were seen in betainf 2-integrin hypomorphic mice backcrossed to the PL/J strain and in betainf 1- integrin transgenic mice. A T cell-based immunopathogenesis of psoriasiform features was shown in a form of graft-versus-host disease in scid/scid mice reconstituted with CD4sup +/CD45RB(hi) T lymphocytes as well as in HLA- B27/hbetainf 2m transgenic rats, demonstrating that dysregulated T cells can induce psoriasiform skin alterations without a primary epithelial abnormality. Finally, xenotransplantation models using human skin grafted on to immunodeficient mice are attractive, as different cell types and some environmental factors leading to psoriasiform features may be studied in human tissue. Overall, although there is no animal model imitating **psoriasis** completely, many aspects of this common human skin disorder are mirrored in the currently available models and psoriatic

6/7/25 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

10620315 EMBASE No: 2000086430
Should a war criminal be rewarded with eponymous distinction?: The double life of Hans Reiter (1881-1969)
Wallace D.J.; Weisman M.
Dr. D.J. Wallace, 8737 Beverly Blvd., Los Angeles, CA 90048 United States
AUTHOR EMAIL: dwallace@ucla.edu
Journal of Clinical Rheumatology (J. CLIN. RHEUMATOL.) (United States) 2000, 6/1 (49-54)
CODEN: JCRHF ISSN: 1076-1608
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 36

The combination of arthritis, urethritis, conjunctivitis, often associated with a psoriasiform rash has been termed Reiter's syndrome, particularly if the patient is HLA-B27 positive. In this report, the history of 'Reiter's syndrome' is investigated by reviewing descriptions of the syndrome and the life and times of Hans Reiter from source materials and recent publications that shed new light on medicine and clinical research in the Nazi regime. The description of the syndrome clearly antedates Reiter's work by several hundred years. Numerous other investigators accurately described a reactive cutaneo-arthropathy before Reiter. Hans Reiter gave his imprimatur to some of the most horrific experiments conducted on concentration camp internees during World War II. We conclude that Reiter does not deserve eponymous distinction. The disorder should be renamed 'reactive cutaneo-arthropathy,' or a 'reactive

10837811 EMBASE No: 2000318499
CTLA4-Ig: A novel immunosuppressive agent
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LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 82

Activation of naive T-cells requires two signals: one is antigen-specific and based on T-cell receptor (TCR) recognition of a peptide-MHC complex and the second is antigen-nonspecific and delivered by specific T-cell receptors after ligation with their ligands (costimulatory molecules) expressed by antigen-presenting cells (APCs). Engagement of the **B7** family of molecules on APCs with their T-cell associated ligands, CD28 and CTLA-4 (CD152), provides a pivotal costimulatory signal in T-cell activation. The lack of costimulation after engagement of the T-cell receptor by antigen, results in a state of antigen-specific unresponsiveness, termed anergy. Manipulation of CD28/**B7** pathway has therefore been envisioned as a potential strategy for achieving therapeutically useful immunosuppression or tolerance. CTLA4-Ig has been initially developed by Bristol-Myers Squibb as a competitive inhibitor of CD28/**B7** pathway (BMS-188667). Thereafter, CTLA4-Ig was produced by Repligen and also in some individual laboratories. In various animal models, discussed in this paper, CTLA4-Ig has been shown to inhibit T-cell-dependent antibody responses, significantly prolong transplanted organ survival, induce long-term donor-specific tolerance in some models, slow progression of autoimmune disease and to have immunomodulatory function in several other immunological disease models. Recently, CTLA4-Ig has entered Phase I clinical trials for the treatment of **psoriasis**, a T-cell mediated skin disease and treatment of graft-versus-host disease in allogeneic bone marrow transplantation. Large clinical randomised trials on the use of CTLA4-Ig are missing, nevertheless, its immunosuppressive effects coupled with features such as specificity of interaction and low toxicity, make CTLA4-Ig a promising new therapeutic agent for induction of donor-specific immunological tolerance, the ultimate goal of clinical

6/7/15 (Item 15 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

08513414 BIOSIS NO.: 199344063414
Discordant expression of CD28 ligands BB-1 and **B7** on keratinocytes in vitro and psoriatic cells in vivo.
AUTHOR: Nickoloff B; Mitra R; Lee K; Turka L; Green J; Thompson C; Shimizu Y
AUTHOR ADDRESS: Dep. Pathol., Univ. Mich., Ann Arbor, Mich.**USA
JOURNAL: Journal of Cutaneous Pathology 19 (6):p543 1992
CONFERENCE/MEETING: 30th Annual Meeting of the American Society of Dermatopathology San Francisco, California, USA December 2-4, 1992
ISSN: 0303-6987
RECORD TYPE: Citation
LANGUAGE: English

69 BIOSIS NO.: 199395116620
Accessory cell function of keratinocytes for superantigens: Dependence on lymphocyte function-associated antigen-1/intercellular adhesion molecule-1 interaction.
AUTHOR: Nickoloff Brian J(a); Mitra Raj S; Green Jonathan; Zheng Xiang-Guang; Shimizu Yoji; Thompson Craig; Turka Laurence A
AUTHOR ADDRESS: (a)Dep. Pathol., Univ. Michigan, Med. Sch. Med., M4232 Med. Sci. I, 1301 Catherine St., Ann Arbor,
JOURNAL: Journal of Immunology 150 (6):p2148-2159 1993
ISSN: 0022-1767
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A growing body of evidence points to a role for epidermal keratinocytes as active participants in immunologic reactions. Inasmuch as certain T cell-mediated skin diseases, such as **psoriasis** and atopic dermatitis, are triggered by microbial infection, we asked whether multipassaged human keratinocytes could provide the costimulatory signals necessary to induce autologous T cell proliferation in response to bacterial-derived superantigens. On exposure to IFN-gamma, keratinocytes are induced to express HLA-DR and HLA-DQ class II MHC Ag, and the lymphocyte-associated Ag-1 counter-receptor intercellular adhesion molecule-1 (ICAM-1). This change in keratinocyte phenotype is accompanied by the ability of these cells to support T cell proliferation induced by two different bacterial-derived superantigens, staphylococcal enterotoxins A and B. Superantigen-driven proliferation in the presence of IFN-gamma-treated keratinocytes was significantly inhibited (70-90% reduction) by mAb against the LFA-1 alpha- or beta-chain or ICAM-1. Proliferation was not inhibited by mAb against the CD28 ligands BB-1 or **B7**, even though these keratinocytes express BB-1. In addition to previous defined roles for class II MHC Ag, stimulation of LFA-1 on the T cells by ICAM-1 on the keratinocytes also plays an important costimulatory role in this superantigen-mediated response. The accessory cell capability of keratinocytes was not unique to superantigen driven responses as PHA, as well as anti-CD3 mAb also induced vigorous T cell proliferation when IFN-gamma-treated keratinocytes were added. However, IFN-gamma-treated keratinocytes consistently failed to provoke an allogeneic response. These data demonstrate that 1) keratinocytes can serve as accessory cells for T cell proliferation using a variety of different stimuli, 2) the LFA-1/ICAM-1 interaction plays a major role in keratinocyte-mediated costimulation, and 3) previous reports in which IFN-gamma-treated keratinocytes failed to support T cell proliferation to nominal or alloantigens, may reflect impaired Ag presentation via class II MHC molecules, rather than lack of necessary costimulatory signals. These findings highlighting the accessory cell function of keratinocytes may have implications for our understanding of the pathogenesis of immunologic disorders of the skin.

09278503 BIOSIS NO.: 199497286873
T lymphocytes in skin lesions of **psoriasis** and mycosis fungoides express **B7-1**: A ligand for CD28.
AUTHOR: Nickoloff Brian J; Nestle Frank O; Zheng Xiang-Guang; Turka Laurence A(a)
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JOURNAL: Blood 83 (9):p2580-2586 1994
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The activation of T cells requires two distinct signals. One signal involves interaction of the antigen-specific T-cell receptor with major histocompatibility complex molecules plus antigenic peptide; a second signal, which is antigen nonspecific, is the interaction of CD28 with its natural ligands **B7-1** and **B7**-2/B70. CD28 is expressed on 80% of T cells, is upregulated after activation, and binds to **B7** gene-family members, found on antigen-presenting cells. Because of our interest in the immunologic basis of benign and malignant T-cell-mediated disorders of the skin, we investigated the cellular distribution of CD28 and **B7** family members in lesions of **psoriasis** and mycosis fungoides. By immunostaining cryostat sections of skin, CD28 was found to be expressed on virtually all lymphocytes in the epidermis and dermis of both skin diseases. Surprisingly, **B7-1** was also found to be expressed on virtually all lymphocytes in the epidermis and dermis of both skin diseases. **B7-1** expression was confirmed on CD3+ T lymphocytes using flow cytometry of single cell suspensions of fresh, unfixed psoriatic lesional tissue. To exclude the possibility that this result was caused by a second reagent contaminating the monoclonal antibody (MoAb) preparation, two different lots were used, and the MoAb was absorbed onto Chinese hamster ovary (CHO) transfectants expressing **B7-1**, or vector-only transfected CHO cells. These procedures confirmed that a **B7-1**-like epitope was being recognized on psoriatic lesional T cells. In contrast to **B7-1** expression on lymphocytes, **B7**-3, as defined by anti-BB-1 MoAb reactivity, was found primarily on epidermal keratinocytes in both skin diseases and was not found on T cells. These results indicate that within two common skin disorders, lesional T cells accumulate in the dermis and epidermis, which express **B7-1**. Such expression may permit self-costimulation involving the CD28-mediated activation pathway, and thereby contribute to the ongoing T-cell proliferation present in these chronic, benign, and malignant skin diseases.

09396113 BIOSIS NO.: 199497404483
Characterization of dermal dendritic cells in **psoriasis**:
Autostimulation of T lymphocytes and induction of Th1 type cytokines.
AUTHOR: Nestle Frank O; Turka Laurence A; Nickoloff Brian J(a)
AUTHOR ADDRESS: (a)Dep. Pathol., Univ. Michigan, M4232 Medical Science I, 1301 Catherine Street, Ann Arbor, MI 4810**USA
JOURNAL: Journal of Clinical Investigation 94 (1):p202-209 1994
ISSN: 0021-9738
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Local activation of T lymphocytes is regarded as an important immunological component of psoriatic skin lesions. Within psoriatic plaques (PP) there are large numbers of dermal dendritic cells (DDCs) immediately beneath the hyperplastic epidermis surrounded by T cells. In this study we investigated the ability of DDCs isolated from PP skin to support immune responses of resting peripheral blood T cells. For comparison, other dendritic cells were obtained from blood of the same psoriatic patients, as well as DDCs from skin of normal healthy individuals (designated NN skin). All dendritic cells studied had high surface expression of HLA-DR, **B7**, and lymphocyte function associated antigen-1 molecules. T cell proliferative responses and cytokine production profiles to these various dendritic cells were measured in the absence and presence of PHA or bacterial-derived superantigens. In the absence of exogenous mitogens, PP skin-derived DDCs were much more effective stimulators of spontaneous T cell proliferation compared with either psoriatic blood-derived or NN skin-derived dendritic cells. Antibody blocking studies revealed involvement of HLA-DR, **B7**, and lymphocyte function associated antigen-1 on PP skin-derived DDCs. Cytokine profiles revealed that in the absence of exogenous stimuli PP skin derived DDCs mediated a T cell response with high levels of IL-2 and IFN-gamma, but not IL-4 or IL-10. NN skin-derived DDCs produced a similar qualitative response, but quantitative amounts of all cytokines measured were lower. Upon addition of PHA or superantigens, both PP skin-derived and NN skin-derived DDCs mediated high levels of IL-2 and IFN-gamma production, with induction of IL-4 particularly evident for PHA reactions. Addition of conditioned medium from psoriatic dermal fragments did not enhance the autostimulatory capacity of blood-derived dendritic cells. These findings highlight the potent autostimulatory potential of PP skin-derived DDCs and suggest an important immunological contribution for these previously overlooked cell types contained within lesional skin

09766436 BIOSIS NO.: 199598221354
Psoriatic skin-derived dendritic cell function is inhibited by exogenous IL-10: Differential modulation of **B7-1** (CD80) and **B7-2** (CD86) expression.
AUTHOR: Mitra Raj S; Judge Thomas A; Nestle Frank O; Turka Laurence A; Nickoloff Brian J(a)
AUTHOR ADDRESS: (a)Dep. Pathol., Univ. Michigan, M4232 Medical Sci. I, 1301 Catherine St., Ann Arbor, MI 48109-0602**USA
JOURNAL: Journal of Immunology 154 (6):p2668-2677 1995
ISSN: 0022-1767
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Regulation of immune responses depends on interactions between APCs and T cells. Such cellular interactions are mediated by surface molecules including MHC class II Ags (DR) and CD28 ligands **B7-1** (CD80) and **B7-2** (CD86). Recent evidence indicates that the presence or absence of costimulatory molecules on APCs significantly influences the qualitative and quantitative nature of an immune response. In this report, we analyze two relevant cytokines in skin immunobiology, granulocyte-macrophage (GM)-CSF and IL-10, and demonstrate their effects on cultured dendritic cells obtained from dermis (DDCs) of normal skin and psoriatic lesions. For comparison, the effects on these professional APCs were contrasted with cultured blood-derived monocytes. Normal and psoriatic skin-derived DDCs express high levels of CD86 over CD80, and the overall hierarchy is DR gt CD86 gt CD80, whereas cultured monocytes express low and equivalent levels of CD80 and CD86. If Ab is added to GM-CSF at the initial period of cultivation, DDCs that emigrate have lower levels of CD86 without any detectable effect on CD80 or DR expression and display a reduced capacity to stimulate either superantigen-driven or alloantigen responsive T cells. Conversely, by adding GM-CSF to monocytes, CD86 levels are enhanced. When IL-10 was added at the beginning of culture, DDCs had significantly lower levels of CD86, without any effect on CD80 or DR expression, and like anti-GM-CSF-treated cells, these DDCs had approximately a 50% reduction in their T cell-stimulating capacity. In contrast, when monocytes were treated identically with exogenously added IL-10, they retained their relatively low levels of CD80 and CD86 with no detectable change in APC function. Blocking studies of DDC:T cell interaction indicated that CD86 was more important than CD80. Thus, differential expression patterns and functional cytokine responses involving these APC populations may be relevant to skin disorders such as **psoriasis**, in which discordant patterns of CD28 ligand expression and disordered cytokine networks are present.

10626520 BIOSIS NO.: 199699247665
CD40 is functionally expressed on human keratinocytes.
AUTHOR: Denfeld Ralf W(a); Hollenbaugh Diane; Fehrenbach Alexandra; Weiss Johannes M; Von Leoprechting Achim; Mai Brigit; Voith Ursula; Schoepf Erwin; Aruffo Alejandro; Simon Jan C
AUTHOR ADDRESS: (a)Dep. Dermatol., Albert-Ludwigs-Universitaet, Hauptstrasse 7, D-79104 Freiburg**Germany
JOURNAL: European Journal of Immunology 26 (10):p2329-2334 1996
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The CD40/gp39 pathway is known to be an important feature of B/T cell collaboration leading to T cell-dependent activation, proliferation or differentiation of B cells. Additionally, CD40 is involved in the regulation of B cell survival and apoptosis. Recently, CD40 has been shown to be expressed functionally on non-hematopoietic cells, i.e. endothelial cells. Here, we demonstrate that human keratinocytes (KC) cultured in vitro express CD40 constitutively. The surface expression of CD40 is markedly up-regulated following stimulation with interferon (IFN)-gamma, but not with tumor necrosis factor-alpha or interleukin (IL)-1-beta. This process is regulated at the CD40 mRNA level as demonstrated by Northern blot analysis. Furthermore, ligation of CD40 via soluble gp39, the CD40 ligand, enhances intercellular adhesion molecule (ICAM)-1 and Bcl-x up-regulation on IFN-gamma-stimulated KC, but not lymphocyte function-associated antigen (LFA)-3, **B7**-2, HLA-DR, or Fas expression. The release of IL-8 is also induced following CD40 ligation on KC. In **psoriasis**, a T cell-mediated inflammatory skin disease, KC have a markedly enhanced expression of CD40. This expression co-localizes with the expression of ICAM-1, Bcl-x, and an influx of CD3+ T cells. These findings suggest a functional role of CD40 on KC in inflammatory skin disorders such as **psoriasis** and could make a therapeutic intervention by disrupting the CD40/gp39 pathway an approach to consider in these inflammatory skin diseases.

11023952 BIOSIS NO.: 199799645097
Functional CD86 (**B7**-2/B70) is predominantly expressed on Langerhans cells in atopic dermatitis.
AUTHOR: Ohki O(a); Yokozeki H(a); Katayama I(a); Umeda T(a); Azuma M; Okumura K; Nishioka K(a)
AUTHOR ADDRESS: (a)Dep. Dermatol., Tokyo Med. Dent. Univ. Sch. Med., 5-45 Yushima 1-chome, Bunkyo-ku, Tokyo 113**Japan
JOURNAL: British Journal of Dermatology 136 (6):p838-845 1997
ISSN: 0007-0963
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Recently, we reported the functional expression of CD86 on cultured human Langerhans cells derived from normal epidermis. In the present study, we investigated the expression and function of costimulatory molecules in the pathogenesis of atopic dermatitis. In immunohistochemical analysis, CD80 and/or CD86 were detected on dendritic-shaped cells not only in the epidermis but also in the dermis in the inflammatory lesions of atopic dermatitis (n = 12). CD80 was expressed in only five cases (42%), while CD86 was expressed in all cases (100%). These molecules were not detected in normal control subjects (n = 8). In non-lesional skin of atopic dermatitis (n = 4), CD86 but not CD80 was detected in one case. CD8 6 was preferentially induced on dendritic-shaped cells in positive patch test sites to Dermatophagoides pteronyssinus or house dust allergen in atopic dermatitis (n = 4). The CD80- or CD86-positive cells were confirmed as Langerhans cells by double immunostaining using anti-CD1a monoclonal antibody. Neither CD86 nor CD80 was detected on keratinocytes. Similar results of the stronger expression of CD86 over that of CD80 were obtained from **psoriasis** vulgaris (n = 11) and from contact dermatitis (n = 7), although CD8 6 was expressed only in 57% of the contact dermatitis cases. The percentage of Langerhans cells positive for CD86 was higher than for CD80, i.e. 48% compared with 9%, respectively, in the epidermis of lesional skin of atopic dermatitis (n = 8). The expression rate of these molecules on Langerhans cells increased in the dermis. To investigate the function of co-stimulatory molecules on Langerhans cells in atopic dermatitis, we conducted an inhibition test with antibodies. Anti-CD86 monoclonal antibody almost completely inhibited T-cell proliferation stimulated with crude extract of D. pteronyssinus in the presence of epidermal cells as antigen-presenting cells, whereas anti-CD80 monoclonal antibody produced less of an inhibitory effect. These data indicate that CD86 expressed on Langerhans cells may play an important part in the pathogenesis of atopic dermatitis.

6/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11985665 BIOSIS NO.: 199900266184
CTLA4Ig-mediated blockade of T-cell costimulation in patients with **psoriasis** vulgaris.
AUTHOR: Abrams Judith R(a); Lebwohl Mark G; Guzzo Cynthia A; Jegasothy Brian V; Goldfarb Michael T; Goffe Bernard S; Menter Alan; Lowe Nicholas J; Krueger Gerald; Brown Michael J; Weiner Russell S; Birkhofer Martin J; Warner Garvin L; Berry Karen K; Linsley Peter S; Krueger James G; Ochs Hans D; Kelley Susan L; Kang Sewon
AUTHOR ADDRESS: (a)Bristol-Myers Squibb Pharmaceutical Research Institute, 5 Research Parkway, Wallingford, CT, 064**USA
JOURNAL: Journal of Clinical Investigation 103 (9):p1243-1252 May, 1999
ISSN: 0021-9738
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Engagement of the **B7** family of molecules on antigen-presenting cells with their T cell-associated ligands, CD28 and CD152 (cytotoxic T lymphocyte-associated antigen-4 (CTLA-4)), provides a pivotal costimulatory signal in T-cell activation. We investigated the role of the CD28/CD152 pathway in **psoriasis** in a 26-week, phase I, open-label dose-escalation study. The importance of this pathway in the generation of humoral immune responses to T cell-dependent neoantigens, bacteriophage phiX174 and keyhole limpet hemocyanin, was also evaluated. Forty-three patients with stable **psoriasis** vulgaris received 4 infusions of the soluble chimeric protein CTLA4Ig (BMS-188667). Forty-six percent of all study patients achieved a 50% or greater sustained improvement in clinical disease activity, with progressively greater effects observed in the highest-dosing cohorts. Improvement in these patients was associated with quantitative reduction in epidermal hyperplasia, which correlated with quantitative reduction in skin-infiltrating T cells. No markedly increased rate of intralesional T-cell apoptosis was identified, suggesting that the decreased number of lesional T cells was probably likely attributable to an inhibition of T-cell proliferation, T-cell recruitment, and/or apoptosis of antigen-specific T cells at extralesional sites. Altered antibody responses to T cell-dependent neoantigens were observed, but immunologic tolerance to these antigens was not demonstrated. This study illustrates the importance of the CD28/CD152 pathway in the pathogenesis of **psoriasis** and suggests a potential therapeutic use for this novel immunomodulatory approach in an array of T cell-mediated diseases.

6/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12465656 BIOSIS NO.: 200000219158
Results of a single-dose, dose-escalating trial of an anti-**B7.1** monoclonal antibody (IDEC-114) in patients with **psoriasis.**
AUTHOR: Gottlieb A(a); Abdulghani A; Totoritis M; Lizambri R; Shuey S; Romano P; Oh C; Chaudhari U; Lebwohl M
AUTHOR ADDRESS: (a)UMDNJ-RWJMS, New Brunswick, NJ**USA
JOURNAL: Journal of Investigative Dermatology 114 (4):p840 April, 2000
CONFERENCE/MEETING: 61st Annual Meeting of the Society for Investigative Dermatology. Chicago, Illinois, USA May 10-14, 2000
ISSN: 0022-202X
RECORD TYPE: Citation
LANGUAGE: English

6/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12552653 BIOSIS NO.: 200000306155
The role of co-stimulation in airway inflammation.
AUTHOR: Djukanovic R
AUTHOR ADDRESS: (a)University of Medicine, Southampton General Hospital, Tremona Road, Level D, Centre Block, Southampton, SO16 6YD**UK
JOURNAL: Clinical and Experimental Allergy 30 (Supplement 1):p46-50 June, 2000
MEDIUM: print
ISSN: 0954-7894
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: There is considerable evidence to support an important role for co-stimulatory molecules in regulating the proliferation and activation of T cells in the immune response. Of particular relevance is the interaction between CD28 on T cells and **B7** expressed on the surface of antigen presenting cells (APCs). CTLA-4, another molecule present on activated T cells may downregulate T cell activity, but its role remains uncertain. CTLA4-Ig, a fusion protein consisting of the extracellular domain of CTLA4 and the Fc portion of human immunoglobulin G1 (IgG1), has been useful for studying the role of CD28/**B7** interactions in immune responses. A number of studies have shown that CTLA4-Ig can switch off T cell activation. In an ovalbumin sensitive murine model of asthma, CTLA4-Ig treatment suppressed the response to inhaled allergen (increased airway hyperresponsiveness (AHR), IgE production, recruitment of eosinophils into the lungs, production of IL-4, IL-5, and IL-10 and increased IFNgamma production from CD3-TCR-activated T cells). Anti **B7**-2 treatment has similar effects suggesting that interaction of **B7**-2 with CD28 is important in the development of a Th-2 type inflammatory response in mice. Recent observations have been of relevance to human allergic disease. In vitro studies have shown that CTLA4-Ig or anti-**B7**-2 antibody can inhibit allergen-induced proliferation and cytokine production by peripheral blood mononuclear cells from atopic subjects. The role of co-stimulation has been studied in a human bronchial explant model of asthma. CTLA4-Ig fusion protein effectively blocked allergen-induced production of IL-5 and IL-13 in bronchial explants from atopic asthmatics. These studies confirm the requirement for interaction between co-stimulatory molecules in cytokine production and allergic inflammation, and point to the CD28-**B7** pathway as being important to the allergen-induced inflammation in asthma. Studies of organ transplantation in primates suggest that CTLA4-Ig is extremely effective in preventing organ rejection. While phase 1 clinical trials have shown CTLA-4-Ig treatment of patients with **psoriasis** vulgaris to be well tolerated and to result in clinical improvement, its role in asthma management merits further investigation.

6/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12465656 BIOSIS NO.: 200000219158
Results of a single-dose, dose-escalating trial of an anti-**B7.1** monoclonal antibody (IDEC-114) in patients with **psoriasis**.

AUTHOR: Gottlieb A(a); Abdulghani A; Totoritis M; Lizambri R; Shuey S; Romano P; Oh C; Chaudhari U; Lebwohl M
AUTHOR ADDRESS: (a)UMDNJ-RWJMS, New Brunswick, NJ**USA
JOURNAL: Journal of Investigative Dermatology 114 (4):p840 April, 2000
CONFERENCE/MEETING: 61st Annual Meeting of the Society for Investigative Dermatology. Chicago, Illinois, USA May 10-14, 2000
ISSN: 0022-202X
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LANGUAGE: English

6/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12666545 BIOSIS NO.: 200000420047
Blockade of T lymphocyte costimulation with cytotoxic T lymphocyte-associated antigen 4-immunoglobulin (CTLA4Ig) reverses the cellular pathology of psoriatic plaques, including the activation of keratinocytes, dendritic cells, and endothelial cells.
AUTHOR: Abrams Judith R(a); Kelley Susan L; Hayes Elizabeth; Kikuchi Toyoko ; Brown Michael J; Kang Sewon; Lebwohl Mark G; Guzzo Cynthia A; Jegasothy Brian V; Linsley Peter S; Krueger James G
AUTHOR ADDRESS: (a)Novartis Pharmaceuticals Corporation, 59 Route 10, Bldg. 122, Rm. S320, East Hanover, NJ, 07936-1080**USA
JOURNAL: Journal of Experimental Medicine 192 (5):p681-693 September 4, 2000
MEDIUM: print
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Efficient T cell activation is dependent on the intimate contact between antigen-presenting cells (APCs) and T cells. The engagement of the **B7** family of molecules on APCs with CD28 and CD152 (cytotoxic T lymphocyte-associated antigen 4 (CTLA-4)) receptors on T cells delivers costimulatory signal(s) important in T cell activation. We investigated the dependence of pathologic cellular activation in psoriatic plaques on **B7**-mediated T cell costimulation. Patients with **psoriasis** vulgaris received four intravenous infusions of the soluble chimeric protein CTLA4Ig (BMS-188667) in a 26-wk, phase I, open label dose escalation study. Clinical improvement was associated with reduced cellular activation of lesional T cells, keratinocytes, dendritic cells (DCs), and vascular endothelium. Expression of CD40, CD54, and major histocompatibility complex (MHC) class II HLA-DR antigens by lesional keratinocytes was markedly reduced in serial biopsy specimens. Concurrent reductions in **B7-1** (CD80), **B7**-2 (CD86), CD40, MHC class II, CD83, DC-lysosomal-associated membrane glycoprotein (DC-LAMP), and CD11c expression were detected on lesional DCs, which also decreased in number within lesional biopsies. Skin explant experiments suggested that these alterations in activated or mature DCs were not the result of direct toxicity of CTLA4Ig for DCs. Decreased lesional vascular ectasia and tortuosity were also observed and were accompanied by reduced presence of E-selectin, P-selectin, and CD54 on vascular endothelium. This study highlights the critical and proximal role of T cell activation through the **B7**-CD28/CD152 costimulatory pathway in maintaining the pathology of **psoriasis**, including the newly recognized accumulation of mature DCs in the epidermis.